Chapter 7

CIGARETTE SMOKE AND MOVING SOURCES OF CARBON MONOXIDE

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The carboxyhemoglobin contained in the blood of smokers originates, in part, from the carbon monoxide emitted by vehicular engines. The proportion of the total carboxyhemoglobin originating from moving sources of carbon monoxide varies from individual to individual depending on the extent of exposure. Those who are inside the moving vehicle are likely to be exposed to the highest concentration of vehicular emission, specially if the vehicle is not properly ventilated with ambient air and the inflow into the vehicle contains a high concentration of the engine exhaust. Workers in garages and tunnels, although outside the vehicle, are in an environment that is partly enclosed preventing free elimination of the vehicular emission into the ambient air. The exposure levels of carbon monoxide of garage men and tunnel inspectors are higher than those of policemen and pedestrians in open areas such as street intersections during hours of heavy traffic .

Smokers with carboxyhemoglobin levels originating from exposure to vehicular carbon monoxide have been compared with nonsmokers. The differences in their health status have been explained by cigarette smoking alone although other variables have not been controlled. The extent of exposure to vehicular traffic is difficult to match between smokers and nonsmokers. The composition of vehicular emission is so complex that a comparable exposure even for a specified group of workers is difficult to attain. The concentration of sulfur dioxide, nitrogen oxides, carbon monoxide and particulate matter vary continuously at various locations and in the same location at different times

of the day. Smokers and nonsmokers exposed to vehicular emissions are likely to suffer from the involuntary inhalation of pollutants.

A. Exposure Inside Vehicles

Most studies relate to automobiles, although there are a few on buses, moving trains, ferry boats and air planes. It is not practicable to apply results from one vehicle to other forms of vehicles because of the differences in size of the engine, the number of passengers and the degree of ventilation.

1. Carbon Monoxide Levels Inside Vehicles.

a. Automobiles. The concentration of carbon monoxide inside motor vehicles has been reported from various states and countries. Goldsmith and Rogers[1] reported the levels inside the driver's compartments in 1,105 California vehicles. Although 92% of the vehicles had levels below 50 ppm, the remainder had concentrations of 50 to 650 ppm. Brice and Roesler[2] reported the following half-hour integrated samples at the following cities:

	No. of Vehicles	Carbon Monoxide ppm mean (Range)
Chicago	16	37 (20 - 50)
Cincinnati	145	21 (8 - 50)
Denver	28	40 (22 - 72)
St. Louis	47	36 (11 - 77)
Washington	44	25 (7 - 43)

In all cities, 75% of the samples taken inside moving vehicles were at least 10 ppm CO and 10% of the samples contained at least 31 ppm CO.

b. Buses. Bulbuyan and Smulevich[3] analyzed the air inside 60 buses. The samples were taken over 2 hours. The mean levels during warm and cold season are as follows:

	mean mg/cu m (ppm)	
	warm season (ppm)	cold season (ppm)
carbon monoxide	42.3	56.5
hydrocarbons	177.2	216
nitrogen oxides	1.5	4.2
gasoline	166.8	114.4

The high pollution levels inside the buses during the cold season were due to engine emissions entering poorly-ventilated buses with windows usually kept shut.

Johnson et al.[4] examined the carbon monoxide levels in school buses in the Seattle area, following an incident in which eight children became ill from carbon monoxide in a school bus. The buses were examined after an estimated driving time of one to two hours, and after idling for two hours at a parking lot.

Concentration CO inside bus	% of total buses after driving 1-2 hrs.	after idling 2 hrs
0	52%	31%
trace to 8 ppm	36%	32%
9-34 ppm	9%	26%
35-49 ppm	3%	5%
50-100 ppm	--	--

From the above results, several buses exceed 35 ppm of carbon monoxide, whether in running or idling conditions.

2. Carboxyhemoglobin Blood Levels of Motor-vehicle Drivers.

As early as 1958, Moureu compared the blood levels of various population groups in France, warning that the blood of drivers of motor vehicles contain, on the average, more carbon monoxide than that of workers exposed to stationary sources of carbon monoxide and to urban residents. The results for 1963 are as follows:[5]

Exposure groups	% Who Have COHb Levels > 4.8%
motor vehicle drivers	41%
workers exposed to CO	14%
urban residents	11%

The high level of carbon monoxide emitted from vehicles in Paris, reflected by elevated carboxyhemoglobin percentages has also been reported by Chovin in 1967.[6] The median for drivers was 5.0% carboxyhemoglobin, whereas the median for policemen was 2.0%. Boudene et al., in 1974, compared the carbon monoxide content of Parisian taxi drivers with a control group of residents.[7] The carbon monoxide content in the blood of drivers was higher than the controls. The comparison included the smoking habits of the drivers but there were no corresponding smokers among the controls so that it was not possible to contrast cigarette smoking alone with exposure to vehicular emission.

An elevation of carboxyhemoglobin levels has been reported among London taxi drivers,[8] Belgrade drivers[9] and Amsterdam drivers.[10] In all these reports, there is a definite elevation of carboxyhemoglobin in motor vehicular drivers compared to non-drivers residing in the same city. Only the comparison of London taxi drivers by Jones et al.[8] has separated smokers from nonsmokers with the following results:

London taxi drivers (No.)	Mean ± S. D.	(Range)
smokers (29)	5.31 ± 2.6	(1.0-4.7)
nonsmokers (20)	1.55 ± 0.8	(0.4-3.0)

As in other comparisons, (Chapter 2), there is overlapping in blood levels between the two groups, and the standard deviation for smokers is larger than that for nonsmokers.

3. Cigarette Smoke Inside Vehicles.

There have been reports of levels of cigarette smoke inside vehicles, believed to be high enough to be of a health hazard to the occupants. The studies have been performed on nonventilated vehicles, a feature that is usually ignored when results are cited by others.

a. Passenger cars. In 1967, Srch[11] placed four subjects in a car with windows and doors closed. The engine was started with the car in the garage. The two smokers consumed 10 cigarettes in 62 min

and analysis of the blood samples revealed the following:

	Mean % COHb		
	before	after	Δ
2 smokers	5	10	5
2 nonsmckers	2	5	3

The increase in carboxyhemoglobin levels in nonsmokers is brought about by carbon monoxide generated from the engine.

It is claimed by Srch[11] that cigarette smoke was the primary source of carbon monoxide but he did not separate vehicular emission from cigarette smoke.

Petrilli et al., reported carboxyhemoglobin levels inside cars with no smokers and with smokers.[11a] Only the minimum values for nonsmokers and the maximum values for smokers were reported, but the exact range for the groups were not mentioned. In any event, the reported minimum for nonsmoker and maximum for smokers were elevated as the carbon monoxide concentration in the car was raised from 50-90, to 60-120 ppm.

The most complete study of the effects of cigarette smoke on carbon monoxide level in a passenger car was performed by Harke and his collaborators[12, 13] in 1975. In an automobile stationed inside a wind tunnel, there were 4 passengers with the driver as a smoker. The windows and doors were closed, but the air jets and air blower were controlled. The carbon monoxide levels at various wind velocity, ventilation and cigarettes

consumed were as follows:[12]

8-10 ppm = 2 x 3 cigarettes, air vents fully open, wind 0 km/hr.

10 ppm = 2 x 3 cigarettes, air vents fully open, wind 50 km/hr.

20 ppm = 2 x 3 cigarettes, air vents 1/2 open, wind 50 km/hr.

30 ppm = 3 x 3 cigarettes, air vents closed, wind 50 km/hr.

The above results indicate the importance of air exchange in determing the level of carbon monoxide from cigarette smoke inside a car. In the absence of ventilation, there is a rise of carbon monoxide concentration, a condition that is rarely reproduced in a real life situation.

The next group of measurements were carried out in automobiles driven through Hamburg. Under the condition of 2 x 2 cigarettes, doors and windows closed, the carbon monoxide levels inside the driven car were as follows:[13]

21 ppm = air vents closed, speed constant

15 ppm = air vents half open, speed constant

12 ppm = air vents open, speed constant

12 ppm = speed 80 km/hr, vents closed

24 ppm = speed 35 km/hr, vents closed

The importance of speed of the car and degree of ventilation is seen in the above results. Air ventilation and car movement reduce the concentration of carbon monoxide.

Normally, comfort dictates a degree of ventilation which minimizes carbon monoxide levels from cigarette smoking. One must also be conscious of the variation in the quality of ventilation systems between the more luxurious American-manufactured cars and many of the foreign compacts.

b. Buses. Seiff conducted a study for the Federal Highway Administration to determine the levels of carbon monoxide inside a Greyhound bus that was stationary.[14] Cigarettes were burning to simulate two conditions: "worst case" condition with all passengers smoking, each one smoking half of the time; the "real world" condition in which the rear fifteen of the seating space occupied by smokers, each one smoking half of the time. In the "worst case" situation, the level of carbon monoxide was 33 ppm, with an ambient air level of 7 ppm, a difference of 26 ppm. The contribution of carbon monoxide from vehicular engine, was not measured separately from the cigarette smoke. In the "real world" situation, the level inside the bus was 18 ppm, ambient of 13 ppm and a difference of 5 ppm. It was the opinion of Seiff that these levels of carbon monoxide were below the standards set by the Occupational Safety and Health Administration.

c. Trains. Harmsen and Effenberger[15] monitored the quality of air inside train cars that were running in Hamburg in 1953. All windows and air vents were closed. Although smoking was permitted there was no increase of carbon monoxide level after 30 to 60 min of use. In one car, with 10 smokers, there was a rise of nicotine content to 2.5 mg/1000 liters, after 45 min. The significance of nicotine level is discussed in Chapter 9.

d. Ferryboat. Godin et al.[16] compared the carbon monoxide concentra-

tions of compartments of a crowded Ward's Island ferryboat:

	CO ppm mean ± S. D. of 11 determinations
smoking compartment	18.4 ± 8.7
nonsmoking compartment	3.0 ± 2.4
difference	15.4 ± 8.2

The levels in the smoking compartment are still below the standard for occupational exposure.

e. Airplanes. The Federal Aviation Administration sampled the air during 20 international flights and 8 domestic flights of the Military Aircraft Command. In all samples, the carbon monoxide level did not exceed 5 ppm.[17]

4. Practical Aspects of Intravehicular Exposures.

The possibility that carbon monoxide contained in cigarette smoke would influence the nonsmoker passenger of a vehicle can be dismissed because the levels even in nonventilated vehicles are below the occupational standards for carbon monoxide. The question as to the influence of smoking on the driver can be answered by citing the investigation of Boek on automobile accidents.[18] There was no difference in accident involvement between smokers and nonsmokers noted by Boek, although a survey of workers by Fiandaca and Vercellotti[19] revealed a correlation between accident rate and exposure to

carbon monoxide in the work environment. Motor vehicular accidents in the city of Los Angeles is not correlated with atmospheric carbon monoxide concentration, but there is an association of the number of accidents to the levels of oxidants in the ambient air.[20] The relationship may be a spurious one reflecting an increase in accidents resulting from heavier traffic, rather than the high level of pollutants causing more accidents. The fatalities from carbon monoxide poisoning inside vehicles[21] are extreme examples which do not relate to normal use of vehicles that are properly ventilated.

B. Extravehicular Exposure in Semienclosed Space

Workers of garages and tunnels are exposed to vehicular emissions that are not readily eliminated. The studies on smokers indicate that the amount of carbon monoxide from smoking is small compared to that emitted from vehicles.

1. Garage Operators.

The first examination of garage operators was reported by Hofreuter et al. in 1962.[22] In a group of vehicular inspectors in Cincinnati, they reported the following values for carboxyhemoglobin % saturation:

	Mean ± S.D. (Range)
garage workers (68):	3.74 ± 1.89 (1.0 - 12.0)
smokers (54)	3.8
nonsmokers (14)	3.4

From the above, Hofreuter et al. concluded that the use of cigarettes was not reflected in a sustained difference between smokers and nonsmokers.

Other investigators have noted a difference between smokers and nonsmokers among garage and service station operators.[23-26] The results of Buchwald[23] from operators in Canada are as follows:

Amount smoked daily (no. of persons)	COHb % saturation Mean (Range) Median
nil (122)	5.0 (0 - 16) 4
1 - 9 (21)	6.4 (1 - 15) 4
10 - 20 (138)	8.5 (0.5-19) 8
>20	9.2 (1 - 18) 9

There is overlapping in the range of values for nonsmokers and smokers, although the means and medians are progressively higher with the amount of cigarettes smoked.

The most significant observations relate to the examination of garage employees, before and after a work shift.[27-30] Ramsey[27,28] reported the

following changes in carboxyhemoglobin levels in a group of 38 parking garage workers:

Time blood collected	Mean ± S.D. (Range)
8:00 A.M.	2.4 ± 1.74 (0.4 - 6.9)
5:00 P.M.	8.4 ± 3.33 (2.9 - 15.8)

The exposure of the workers was estimated to be 59 ppm of carbon monoxide in the work environment. Out of the 38 employees, the breakdown of smokers and nonsmokers were as follows:[27]

Time of collection	Mean ± S.D. for COHb %	
	24 Smokers	14 Nonsmokers
8:00 A.M.	2.9 ± 1.88	1.5 ± 0.83
5:00 P.M.	9.3 ± 3.16	7.3 ± 3.46
difference	6.4	5.8

The most significant feature of the above results is that the uptake of carbon monoxide during a working day (about 6%) is four times higher than the difference between smokers and nonsmokers (1.5%). The amount of carbon monoxide absorbed from smoking is small compared to the amount from exposure to vehicular emission in a garage. Ramsey[28] has further described a reduction in reaction times in drivers exposed to carbon monoxide levels seen in garage operators.

2. Longshoremen.

In the course of operating fork lift trucks in ship holds or unloading of cars, longshoremen are exposed to carbon monoxide emitted from vehicular motors.[31-34] Smokers absorb less carbon monoxide from the ship holds than nonsmokers.[31] Gibbs et al.

blood samples collected:	Median (Range) for COHb % 39 smokers	12 nonsmokers
arrival at work	3.4 (0.5 - 11.2)	0.6 (0 - 1.6)
end of first period	3.4 (0.0 - 8.3)	2.0 (0 - 5.0)
difference	0	1.4

The phenomenon of no uptake of carbon monoxide seen in smokers is explained by their level of carboxyhemoglobin upon their arrival. The level of 3.4% is enough to reduce the absorption of carbon monoxide from their work environment (see Chapter 1).

3. Tunnel Traffic Officers.

The concentration of carbon monoxide in tunnels vary according to the density of vehicular traffic and velocity of air flow.[35] Although the levels can reach as high as 250 ppm, the concentration is usually below 100 ppm.[36] An examination of a group of 156 Holland Tunnel traffic officers exposed throughout a period of 13 years to an occupational level averaging 70 ppm did not reveal any evidence of injury to health attributable to carbon monoxide.[37]

In a more recent health examination of the employees of the New York City Tunnel authority exposed to an average level of 63 ppm, the carboxyhemoglobin levels were as follows: 2.93 ± 1.36% for nonsmokers and 5.01 ± 2.25% for smokers.[38] As in other comparative groups, the standard deviation for smokers is larger than in nonsmokers (Chapter 2). There was no influence of smoking in ventilatory function. Although the closing volume was higher in both smokers and nonsmokers compared to hospital maintenance workers, carbon monoxide exposure cannot explain the difference because the air inside the tunnel also contained significant amounts of lead, nickel, hydrocarbons, nitrogen oxides, aldehydes and acrolein.[38]

C. Inhalational Toxicity of Vehicular Exhaust Gas

Since carboxyhemoglobin levels of passengers and vehicular drivers are determined by the amount of carbon monoxide emitted by the engine, it is important to review the effects of total vehicular exhaust gas. In 1921, Hendersen et al. reported the physiological effects of automobile exhaust gas on human volunteers in a chamber.[39] The standards of ventilation for brief exposure to carbon monoxide were based on the concentrations that did not elicit dizziness, headache and nausea, the first perceptible signs of carbon monoxide intoxication. Repeated daily exposures to 200, 300 and 400 ppm, 4 to 7 hours daily for 68 days were performed on a group of human volunteers by Sayers et al. in 1929.[40] Four hours of exposure

to 200 ppm caused frontal headache to appear in some subjects, with a level of 20% carboxyhemoglobin saturation. Following the report of Henderson et al. there were studies of toxicity of exhaust gases and carbon monoxide in air mixtures. The limited number of investigations using exhaust gas per se are discussed in the remainder of this chapter.

1. Humans.

Holland et al.[41] exposed students in a chamber containing exhaust gas with 15 to 29 ppm of carbon monoxide. After a few hours of exposure, there was no alteration in the performance of fine neuromuscular tasks, e.g. reaction time or cardiorespiratory work efficiency. There have been no other reported studies on humans exposed in chamber containing exhaust gas.

2. Dogs.

Vaughan et al. exposed 104 beagles for 18 months to natural and photochemically reacted auto exhaust gas.[42] The concentration of carbon monoxide was 100 ppm, with 24 to 30 ppm of hydrocarbons, 0.1 ppm of nitrogen dioxide and 1.5 - 2.0 ppm of nitrogen oxide. There were no differences in diffusing capacity, pulmonary compliance, and pulmonary resistance between exposed and control dogs. The other investigations used individual constituents of exhaust gas rather than the total mixture[43,44] so that there is no information on the threshold concentration of exhaust gas that would influence the canine lungs when administered for prolonged periods.

3. Rabbits.

The exhaust gas used on rabbits contained as much as 1,000 ppm CO which caused a temporary increase in dust elimination capacity of the trachea and later a decrease.[45] In the other exposure studies, lower concentrations of exhaust gas (30 ppm CO) were used but there were no measurements of lung function.[46,47]

4. Guinea Pigs.

The most widely used species for inhalational toxicity is the guinea pig. Swan and Balchum[48] maintained animals for prolonged periods and noted an increase in airway resistance when the ambient air exceeded the following levels: 40 ppm carbon monoxide, 16 ppm hydrocarbons, 1.2 ppm nitrogen oxides and 0.2 ppm oxidants. In anesthetized guinea pigs, each of the pollutants has been demonstrated to depress the myocardium and constrict or dilate the airways.[50,51]

5. Rats and Mice.

The experiments on rats and mice indicate that chronic exposure to exhaust gas produce alveolar lesions,[52-54] pulmonary tumors[55-58] and bronchial inflammation.[59] The threshold concentrations reported for the various constituents have been variable indicating differences in susceptibility of strains and in exposure schedule.[60,61] The results so far indicate that the components of exhaust gas, when inhaled produce lung lesions which cannot be duplicated by administration of carbon monoxide.

D. Practical Aspects of Vehicular Exposure.

Although results of animal experiments indicate that vehicular emissions when inhaled produce pulmonary lesions, it has not been possible to ascertain the human threshold levels in man. The level of carboxyhemoglobin in the blood has been used as the tracer for the amount of human exposure to vehicular emission as well as to cigarette smoke. The interpretation of epidemiologic data has been difficult because the effects from cigarette smoking are distorted by the exclusion of those arising from vehicular emission. Studies that recognize the dual source of carbon monoxide have evaluated the influence of cigarette smoking as less important than the exposure to vehicular traffic.

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